INDOOR MOLDS AND NEUROBEHAVIORAL PULMONARY IMPAIRMENT PRELIMINARY REPORT

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I. Introduction

In about 1985 asthma and flu-like symptoms were associated with molds growing indoors (1,2). Previously farmers exposed to mold feed had asthma and hypersensitivity pneumonitis. Broadening farming disorders people exposed indoors complained of odors, memory loss, lack of concentration, sleep disturbances and depression (2,3). Mold growth occurred with water condensation in walls, in air ducts and under carpets, tile and flooring (1). Water had leaked from roofs, windows, water pipes, shower stalls, bath tubs and drains. Mold spores were observed microscopically and grown from air and surface samples (1,2). Likewise, people's serum IgG, IgM, IgE and IgA antibodies to molds appeared elevated and saliva showed elevated IgA antibodies (2). Antibodies to aflatoxins, trichothecenes and satratoxins were frequent (3). Case recognition relied on symptoms and on molds verified microscopically and grown in culture. Respiratory symptoms and complaints of defective memory and concentration, impaired balance and depression (2,3) led to measurements of lung and central nervous system functions in 65 consecutive mold exposed patients in 2001-2002.

II. Methods

A consecutive case series was compared to a referent group. We compared 65 consecutive outpatients *who were* mold exposed in their homes in Arizona, California, and Texas to 202 community subjects without known mold or chemical exposure. We measured balance, choice reaction time, color discrimination, blink reflex, visual fields, grip, hearing, problem solving, verbal recall, perceptual motor speed, and memory using methods described in detail previously (3,4). Medical histories and chemical exposures were recorded on a questionnaire. Mood states were rated in 5 steps from not at all to extremely and of 35 symptom assigned frequencies in 10 intervals from never to always using checklists (4,6). Spirometry measured pulmonary volumes and flows and they were compared to age and sex adjusted predictions (7). Neurobehavioral

comparisons were made after adjusting individual measurements for age, educational attainment, and sex (8). Significance of differences between groups was assessed by analysis of variance, with $p<.05$ considered significant.

III. Results

The mold exposed group compared to referents had impaired balance, reaction time, blink reflex latency, color discrimination, visual fields, and grip (Table 1). Also their scores were reduced for digit symbol substitution, peg-placement, trail making, verbal recall, and picture completion. Twenty-one of 26 tested functions were abnormal. The numbers and pattern of abnormal tests were similar, but exceeded the 5.7 in people exposed to polychlorinated biphenyls, PCBs (4). Exposed people averaged 9.9 abnormalities compared to 2.3 in controls. Mood State scores had a mean of 64 compared to 21 in unexposed and symptom frequencies were elevated at 5.0, similar to the 5.1 in PCB exposed people (4) and nearly twice the frequencies of the unexposed group. (8) Airway obstruction was found, vital capacities reduced, so that the ratio of forced expiratory volume in one second to forced vital capacity increased, Table 2.

IV. Discussion

These patient's complaints suggested mold exposures including persisting flu-like illnesses, onset of asthma, beginning of severe fatigue, impaired memory and concentration, and frequent dizziness and unsteady balance. These were accompanied by musty odors and black mold growth on baseboards, lower walls, particularly of bathrooms, adjoining bedrooms and closets and on outer walls beneath window sashes and where walls joined ceilings. Usually water leaks were found from roofs, windows, outer walls, or within walls. Microscopic examination of touch preparation showed spores and hyphae of many different molds. Serum and saliva antibodies for many molds exceeded laboratory reference values but only Satratoxin antibodies were elevated when compared to an unexposed group (in preparation).

Clinical judgment attributed these impairments to mold after considering other possibilities as these patients were without preexisting medical or neurological diseases or traumatic brain damage. Neither did mold exposed patients have no previous chemical exposures and they had rarely used pesticides.

Pulmonary function abnormalities indicated airways obstruction that reduced vital capacity and FEV_1. The increased FEV_1/FVC ratio was characteristic of bronchiolitis (9,10).

Inhaled mycotoxins, liberated from indoor mold growth, caused brain impairment and neurological symptoms. Trichothecenes are epoxides that covalently adduct DNA, RNA, protein and microtubules of nerve axons (11,12), providing mechanisms for lung, brain, and immune system toxicity.

But why did people develop sickness from mold in their homes in 2000-2002?

We suggest the following sequence. After World War II construction of the interior walls of homes shifted from wood or metal lath, plaster and lime coat to plasterboard (gypsum board) that reduced cost. The inner paper (cellulose) layer of the wall board encouraged mold growth (13,14) when inner walls became damp from inadequate venting of moisture or leaks from walls, roofs, or plumbing (15). Variation in moisture stressed molds to make spores and toxins such as trichothecenes and satratoxin (16,17) that escaped into living and working spaces (3,10). In earlier times alkaline plaster or lime coating on walls discouraged fungal growth.

V. References

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TABLE 1

SUMMARY OF ABNORMALITIES IN 65 MOLD/MYCOTOXIN EXPOSED SUBJECTS (65) TO 202 REFERENT SUBJECTS AND 98 EXPOSED TO PCBs COMPARED AS PERCENT OF PREDICTED, BY ANALYSIS OF VARIANCE

	Mold	*Controls*	*PCPs exposed*
Simple Reaction Time	+	0	+
Choice Reaction Time	+	0	+
Balance Sway Speed			
Eyes Open	+	0	+
Eyes Closed	+	0	+
Blink Reflex Latency R-1 (ms)			
Right	+	0	0
Left	+	0	0
Hearing Losses			
Right	0	0	0
Left	0	0	0
Color Discrimination Errors			
Right	+	0	+
Left	+	0	+
Visual Field Performance			
Right	+	0	+
Left	+	0	+
Grip strength			
Right	+	0	0
Left	+	0	0

Table 1 cont'd:

	Abnormal %	*Controls*	*PCP exposed*
Culture Fair	0	0	+
Digit Symbol	+	0	+
Vocabulary	+	0	+
Verbal Recall			
Immediate	+	0	+
Delayed	+	0	+
Pegboard	+	0	+
Trails A	+	0	+
Trails B	+	0	+
Finger Writing Errors			
Right	0	0	+
Left	0	0	0
Information	+	0	+
Picture Completion	+	0	+
Similarities	0	0	+
Total Abnormalities mean	*9.9*	*2.3*	*5.7*
Total Symptom Frequencies mean	*5.0*	*2.6*	*5.1*
Total POMS mean	*64*	*21*	*73*

TABLE 2

PULMONARY FUNCTIONS, FORCED VITAL CAPACITY, FORED EXPIRATION VOLUME AND THEIR RATIO IN 65 MOLD EXPOSED SUBJECTS COMPARED TO 202 REFERENT SUBJECTS AS PERCENT OF PREDICTED BY ANALYSIS OF VARIANCE.

	65 Exposed Mean	*202 Unexposed Mean*	*P values*
FVC	91.9	101.6	.0001
FEV1	88.4	93.6	.016
FEV1/FVC	77.2	72.9	.0007

Table 3

COMPARISON OF PREVALENCE OF ABNORMAL TITERS, IN PERCENT, TO 3 MYCOTOXINS AND 14 ANTIBODIES TO MOLD AND 54 MOLD EXPOSED PATIENTS WITH SYMPTOMS AND IMPAIRMENTS, AND 48 COMMUNITY (UNEXPOSED) SUBJECTS

	Unexposed Control Pop. (48)	*Mold-exposed (54)*
IgG	52	74 (> 42%)
IgM	44	94 (>114%)
IgA	79	33 (< 42%)
IgE	79	117 (> 47%)
Mycotoxin		
Aflatoxin	16.7	9.2 (<55%)
Satratoxin	22.9	40.7 (>178%)
Trichothecene	16.7	13.0 (<22%)

Mold antibodies measured:
Alternaria tenus, Aspergillus fumigatus, Aspergillus niger, Aspergillus versicolor, Chaetomium globosum, Cladosporium herbarum, Epicoccum nigrum, Geotrichium candidum, Penicillium notatum, Phoma herbarum, Pullullaria pullulans, Rhizopus nigricans, Rhodotorula glutinis, Stachybotrys chartarum, Aflatoxin, Satratoxin, Trichothecenes